

**AMENDMENTS TO THE CLAIMS:**

1. (previously presented) A method of treating a lung proliferative vascular disorder in a patient comprising administering an HMG-CoA reductase inhibitor,

wherein the HMG-CoA reductase inhibitor is present in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient; and

wherein said lung proliferative vascular disorder is selected from the group consisting of primary pulmonary hypertension, secondary pulmonary hypertension, Eisenmenger's syndrome, chronic thromboembolic disease, pulmonary fibrosis, obliterative bronchiolitis, and lymphangioleiomyomatosis.

2. (canceled)

3. (currently amended) ~~The method of claim 1, wherein the lung proliferative vascular disorder is~~ A method of treating primary pulmonary hypertension in a patient comprising:

administering an HMG-CoA reductase inhibitor in a pharmaceutical formulation comprising a pharmaceutically acceptable carrier at a dose of from about 0.1 to about 100 mg/kg per day, wherein the formulation further comprises in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient.

4. (original) The method of claim 1, wherein the HMG-CoA reductase inhibitor is selected from the group consisting of atorvastatin, cerivastatin, fluvastatin, lovastatin, mevastatin, pravastatin, pitastatin, rosuvastatin and simvastatin.

5. (original) The method of claim 1, wherein the HMG-CoA reductase inhibitor is simvastatin.

6. (original) The method of claim 1, wherein the HMG-CoA reductase inhibitor is administered in a pharmaceutical formulation at a dose of from about 0.1 to about 100 mg/kg per day.

7. (original) The method of claim 6, wherein the formulation further comprises a pharmaceutically acceptable carrier suitable for oral, parenteral, transdermal, transmucosal, or pulmonary delivery.

8. (previously presented) The method of claim 1, further comprising administering an additional active agent, wherein said additional active agent is selected from the group consisting of anticoagulants, vasodilators, macrolide anti-inflammatory agents, diterpenoid triepoxides, endothelin receptor antagonists, geranyl transferase inhibitors, farnesyl transferase inhibitors, and inhibitors of EGF tyrosine kinase, and pharmaceutically acceptable salts and esters thereof.

9. (canceled)

10. (previously presented) The method of Claim 8, wherein the additional active agent is a vasodilator selected from the group consisting of prostanoids, phosphodiesterase (PDE) inhibitors, nitric oxide, nitric oxide precursors and calcium channel blockers.

11. (canceled)

12. (original) The method of 10, wherein the prostanoid is prostacyclin, treprostinil, iloprost, beraprost, prostaglandin E<sub>1</sub> or prostaglandin E<sub>2</sub>.

13. (original) The method of claim 12, wherein the prostanoid is prostacyclin.

14-18 (canceled)

19. (previously presented) The method of claim 1, wherein neointimal smooth muscle cell hyperplasia is decreased upon treatment with the HMG-CoA reductase inhibitor, thereby reducing the neointimal smooth muscle cell hyperplasia in the pulmonary arteries of the patient.

20. (previously presented) The method of claim 1, wherein the lung proliferative vascular disorder is characterized by vascular occlusion in the pulmonary arteries of the patient, and wherein the vascular occlusion is reversed upon treatment with the HMG-CoA reductase inhibitor, such that an increase in blood flow is provided through the pulmonary arteries.

21. (original) The method of claim 20, wherein the blood flow is increased by from about 5% to at least about 300%.

22. (previously presented) The method of claim 1, wherein the lung proliferative vascular disorder is characterized by pulmonary hypertension, and wherein the hypertension is reversed upon treatment with the HMG-CoA reductase inhibitor.

23. (currently amended) ~~The method of claim 7,~~ A method of treating a primary pulmonary hypertension in a patient comprising:

administering an HMG-CoA reductase inhibitor in a pharmaceutical formulation comprising a pharmaceutically acceptable carrier suitable for pulmonary delivery at a dose of from about 0.1 to about 100 mg/kg per day, wherein the formulation further comprises in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient;

wherein the HMG-CoA reductase inhibitor is administered by inhalation.

24. (previously presented) The method of claim 23, wherein the HMG-CoA reductase inhibitor is administered using a dry powder inhaler, metered dose inhaler, or nebulizer.

25 – 29 (canceled)

30. (previously presented) A method of reversing right ventricular hypertrophy in a patient suffering from pulmonary hypertension comprising administering an HMG-CoA reductase inhibitor.

31. (canceled)

32. (previously presented) The method of claim 30, further comprising administering an additional active agent selected from the group consisting of anticoagulants, vasodilators, macrolide anti-inflammatory agents, diterpenoid triepoxides, inhibitors of EGF tyrosine kinase receptor signaling, geranyl transferase inhibitors, farnesyl transferase inhibitors, and endothelin receptor antagonists, and pharmaceutically acceptable salts and esters thereof.

33. (currently amended) ~~A method of treating pulmonary hypertension comprising administering~~ The method of Claim 30, wherein the HMG-CoA reductase inhibitor is simvastatin.

34-36. (canceled)

37. (currently amended) ~~The method of claim 33,~~ A method of treating a primary pulmonary hypertension in a patient comprising:

administering simvastatin in a pharmaceutical formulation comprising a pharmaceutically acceptable carrier suitable for pulmonary delivery at a dose of from about 0.1 to about 100 mg/kg per day, wherein the formulation further comprises in an amount effective to reduce vascular occlusion in the pulmonary arteries of the patient, and which does not substantially increase endothelial cell nitric oxide synthase activity in the endothelial cells of the pulmonary arteries of the patient;

wherein the simvastatin is administered by inhalation.

38. (canceled)

39. (previously presented) The method of claim 33, further comprising administering an additional active agent selected from the group consisting of anticoagulants, vasodilators, macrolide anti-inflammatory agents, FK506, inhibitors of EGF tyrosine kinase receptor signaling, diterpenoid triepoxides, geranyl transferase inhibitors, farnesyl transferase inhibitors, and endothelin receptor antagonists, and pharmaceutically acceptable salts and esters thereof.